Biomedical Science

Acetazolamide or Dexamethasone Use Versus Placebo to Prevent Acute Mountain Sickness on Mount Rainier

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Eighteen climbers actively ascended Mount Rainier (elevation 4,392 m) twice during a randomized, double-blind, concurrent, placebo-controlled, crossover trial comparing the use of acetazolamide, 250 mg, dexamethasone, 4 mg, and placebo every 8 hours as prophylaxis for acute mountain sickness. Each subject was randomly assigned to receive placebo during one ascent and one of the active medications during the other ascent. Assessment of acute mountain sickness was performed using the Environmental Symptoms Questionnaire and a clinical interview. At the summit or high point attained above base camp, the use of dexamethasone significantly reduced the incidence of acute mountain sickness and the severity of symptoms. Cerebral and respiratory symptom severity scores for subjects receiving dexamethasone (0.26 ± 0.16 and 0.20 ± 0.19, respectively) were significantly lower than similar scores for both acetazolamide (0.80 ± 0.80 and 1.20 ± 1.05; $P = .025$) and placebo (1.11 ± 1.02 and 1.45 ± 1.27; $P=.025$). Neither the use of dexamethasone nor that of acetazolamide measurably affected other physical or mental aspects. Compared with placebo, dexamethasone appears to be effective for prophylaxis of symptoms associated with acute mountain sickness accompanying rapid ascent. The precise role of dexamethasone for the prophylaxis of acute mountain sickness is not known, but it can be considered for persons without contraindications who are intolerant of acetazolamide, for whom acetazolamide is ineffective, or who must make forced, rapid ascent to high altitude for a short period of time with a guaranteed retreat route.

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Acute mountain sickness (AMS) is a syndrome characterized by headache, nausea, vomiting, insomnia, and lassitude.[1] The syndrome, part of a continuum including high-altitude pulmonary edema[2] and high-altitude cerebral edema,[3] generally occurs in lowlanders three to eight hours after they ascend to an altitude greater than 3,000 m.[4,5] The increased popularity of recreation in mountain settings, along with the greater accessibility brought about by advances in transportation systems, has contributed to the increase in the incidence of AMS. Slow, staged ascent remains the best method of preventing AMS.[6] Interest continues in developing a prophylactic regimen that is rapid acting, effective, and well tolerated.

In the past, much of the interest concerning the chemoprophylaxis of AMS has focused on acetazolamide.[4,7-17] Recent studies, however, have shown the use of dexamethasone to be effective in reducing the incidence and severity of AMS symptoms.[18-22] A previous study comparing these two agents in different cohorts ascending Mount Rainier, an ideal setting for the study of AMS and prophylactic drug regimens under actual climbing conditions,[13,23] showed an acetazolamide-induced side effect (nausea) at low elevations, which confounded comparison of acetazolamide and dexamethasone.[20] In the present study, we have reexamined the efficacy of prophylactic acetazolamide and dexamethasone use relative to placebo during repeated, rapid, active ascents of Mount Rainier using a crossover design.

Patients and Methods

Eighteen climbers made two separate ascents of Mount Rainier scheduled at least two weeks apart. Using a random numbers table, climbers were allocated to receive one of two active chemoprophylactic drugs, dexamethasone and acetazolamide, to be taken during one of their ascents. During the other ascent, they received placebo. The order of active drug or placebo administration was assigned randomly as well. Dosage regimens consisted of either acetazolamide, 250 mg (Geneva Generics, lot 31578); dexamethasone, 4 mg (Roxane, lot 861121); or lactose placebo (MCB Reagents, lot LX0035-3) administered every 8 hours beginning 24 hours before the start of each climb and continuing until descent from the high point. The drugs were packaged in identical-appearing pink capsules by Pharmaceutical Services, University of Washington,* and distributed at orientation and baseline data-gathering sessions several days before each ascent.

The subjects normally resided at sea level and had not been exposed to high altitude within three weeks before the study. All were free of cardiorespiratory disease, and none had a history of diabetes mellitus, sulfa drug allergy, acid peptic disease, or psychiatric illness. The study was approved by the University of Washington Human Subjects Review Committee, and all subjects gave informed consent.

*Theodore Taniguichi, MS, Director of Pharmaceutical Services, Department of Pharmacy, University Hospital, University of Washington, prepared the medication.

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The Camp Schurman Climbing Rangers, Mount Rainier National Park, assisted with this study.

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ABBREVIATIONS USED IN TEXT

AMS = acute mountain sickness
AMS-C = cerebral symptoms of acute mountain sickness
AMS-R = respiratory symptoms of acute mountain sickness
ANOVA = analysis of variance
CLAT = Conceptual Level Analogy Test
ESQ-III = Environmental Symptoms Questionnaire

Subjects drove from sea level to a trailhead at 1,300 m on the northeast side of Mount Rainier. Active ascent to a base camp at approximately 3,000 m took an average of seven hours. The ascent to the summit began early in the morning on the second day, required about seven hours, and was followed by the return to the trailhead the same day. Symptoms of AMS were assessed using the Environmental Symptoms Questionnaire, second revision (ESQ-III), a 67-question symptom inventory designed to quantitate the severity of symptoms (using a 6-point Likert scale) specifically associated with exposure to altitude and other stressful environments.[24] The questionnaire was self-administered by subjects at sea level (24 hours after they began taking the study medications) and during the climb, within 15 minutes of reaching the following points: 1,300 m (trailhead), 3,000 m (base camp) on ascent, 4,392 m (summit) or high point attained above base camp, and 3,000 m (base camp) on descent. Weighted averages of cerebral (AMS-C) and respiratory (AMS-R) symptom scores were calculated. Symptoms such as headache, nausea, and insomnia contributed to the AMS-C score while symptoms such as shortness of breath and rapid heartbeat contributed to the AMS-R score. As suggested by the results of earlier studies, scores of greater than 0.7 for AMS-C and 0.6 for AMS-R were used to indicate the presence of AMS.[24]

Immediately following completion of the questionnaire, a clinical interview and examination were conducted by one of the investigators (A.J.E.) without knowledge of the subjects' responses to the questionnaire. Scoring was based on a scale that assigns 1 point each for headache, insomnia, anorexia, and dizziness; 2 points each for vomiting and headache unrelieved by analgesics; and 3 points for dyspnea at rest, ataxia, and severe lassitude. Scores of 2 or greater are considered diagnostic of AMS.[25,26]

Additional measures of physical and mental function at sea level and at the summit or high point attained included a five-minute Harvard Step Test,[27] the Conceptual Level Analogy Test,[28] and a test of rapid alternating finger movement as an adaptation of the finger oscillation test.[29] Measurements of blood oxygen saturation and heart rate were attempted with a portable pulse oximeter but were unsuccessful because of cold temperatures.*

A split-plot analysis of variance (ANOVA)[30] was used to compare the efficacy of acetazolamide and dexamethasone use against that of placebo and each other. Drug type (placebo vs active chemoprophylaxis or dexamethasone vs acetazolamide) was a fixed categorical covariant. Altitude was a fixed linear covariant. The indices of AMS (AMS-C, AMS-R, and clinical interviews) were used as dependent variables. To check for differences in the two groups of climbers during the placebo climb (a check on the randomization scheme), Mann-Whitney rank sum statistics were computed.[31] Finally, Fisher's exact test[32] was used to evaluate the significance of contingency table analyses.†

*Bill Anton, RRT, Respiratory Therapy, University Hospital, assisted, and Ken Craig, Physio Control, Redmond, Washington, provided the pulse oximeters.

Results

Group comparisons of the 18 subjects based on active drug received (11 men and 7 women, mean age 34.6±9.2 years) are shown in Table 1. All but 2 of the 18 subjects (89%) reached the summit on at least one of their two climbs. These two subjects, both in the acetazolamide group, had AMS symptoms that prevented ascent when using both drug and placebo. Adverse weather conditions were responsible for the other failed summit attempts. The lowest high point attained by subjects completing the study was 3,871 m, and the

†Jim Hughes, MS, provided statistical consultation.

TABLE 1.—*Group Comparisons Based on Active Drug Prophylaxis**

Clinical Characteristic	*Acetazolamide, n=8*	*Dexamethasone, n=10*
Age, yr	32.6±3.9	36.2±2.4
Male, No. (%)	6 (75)	5 (50)
Previous Mt Rainier ascent, No. (%)	3 (38)	3 (30)
Past history of AMS, No. (%)	5 (62)	3 (30)
Time, base camp to high point, h	5.7±0.6	6.6±0.3
Total time to high point, h	24.8±0.0	25.6±0.5
Study drug taken, doses	7.0±0.5	7.1±0.3

AMS = acute mountain sickness

*Mean and standard error of the mean, except as noted.

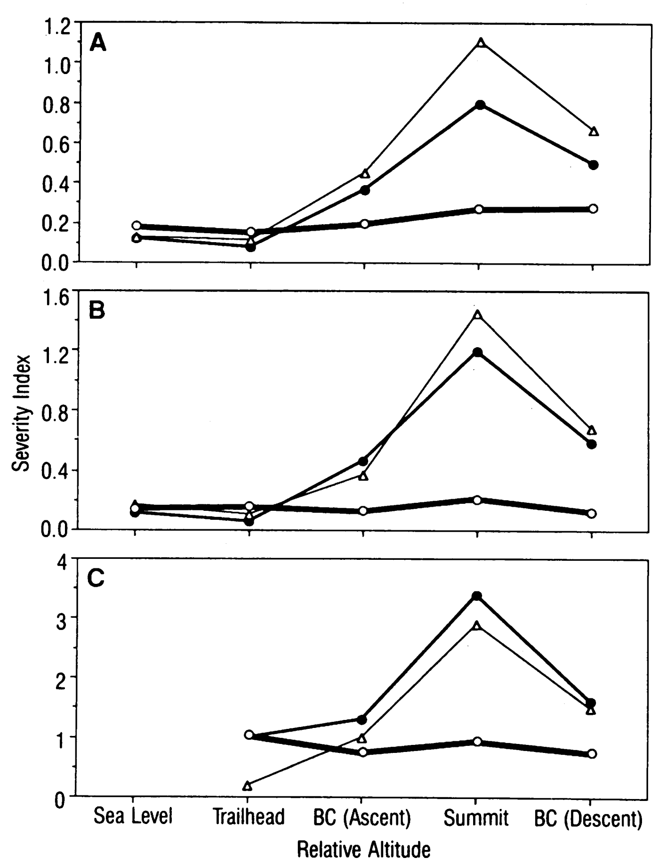

Figure 1.—The graphs show the relative severity of mean **(A)** cerebral symptoms index (AMS-C), **(B)** respiratory symptoms index (AMS-R), and **(C)** total interview scores for subjects receiving active and placebo prophylaxis against acute mountain sickness during a climb of Mount Rainier. BC = base camp, ● = acetazolamide, ○ = dexamethasone, △ = placebo

average time spent ascending from sea level to the high point was 25.2 hours (range, 21.0 to 27.5 hours). Of climbers assigned to receive dexamethasone, 30% had a previous history of AMS, compared with 62% of those receiving acetazolamide ($P = .18$).

At the summit or high point attained, the dexamethasone group had significantly fewer symptoms and a lower incidence of AMS than either the acetazolamide or placebo group as evaluated by both questionnaire and clinical interview (Figure 1). Cerebral symptom scores ($\pm$ standard deviation) at the high point for climbers receiving dexamethasone (0.26 ± 0.16) were significantly lower than those for climbers receiving acetazolamide (0.80 ± 0.80) and placebo (1.11 ± 1.02, $P = .025$). Similarly, AMS-R scores were lower for subjects receiving dexamethasone (0.20 ± 0.19) than for those using acetazolamide (1.20 ± 1.05) and placebo (1.45 ± 1.27, $P < .025$). The difference between the acetazolamide and placebo groups at the highest elevation was not statistically significant.

At the summit or high point (Figure 2), none of the subjects taking dexamethasone were designated "sick" by ESQ-III criteria[23] and only 10% were by clinical interview criteria.[24,25] By contrast, 40% of those taking acetazolamide were "sick" according to the ESQ-III ($P = .02$) and 75% were according to clinical interview ($P = .01$).

Overall, performance on the five-minute Harvard Step Test deteriorated in all groups (decrease from base line to high point: acetazolamide, 31%; dexamethasone, 20%; placebo, 35%) but was not significantly different by group. Similarly, there was little difference in mental function between groups. Conceptual Level Analogy Test (CLAT) scores were higher compared with placebo at the summit for climbers taking dexamethasone (mean score 13.1 ± 2.2 vs placebo 12.3 ± 2.9) and those taking acetazolamide (mean score 14.4 ± 3.1 vs placebo, 13.3 ± 2.8), not a significant difference. Comparison of the decline in CLAT scores by group also revealed no differences during the active drug climb (decline with dexamethasone use 21% vs placebo, 22%; decline with acetazolamide use 14% vs placebo, 20%). Dominant finger oscillation testing at the summit was not significantly different with the use of dexamethasone (55.0 ± 3.6) compared with placebo (49.7 ± 10.7) or receiving acetazolamide (52.0 ± 4.5) compared with placebo (54.0 ± 3.4).

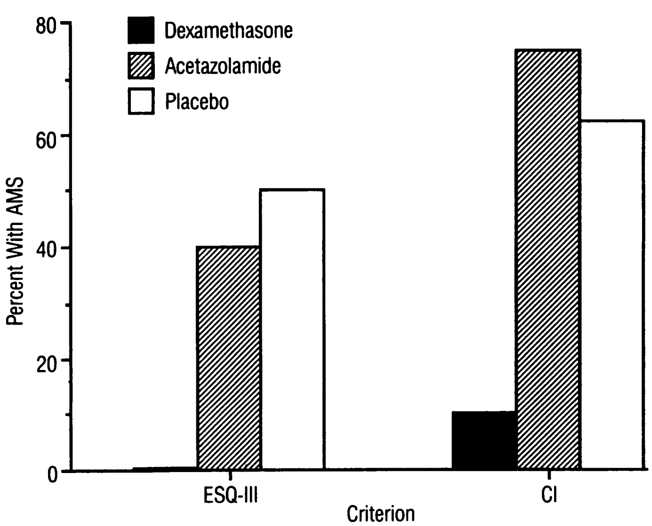

Figure 2.—A significant difference was found in the incidence of acute mountain sickness (AMS) at the summit or high point on Mount Rainier in subjects receiving prophylaxis with dexamethasone, acetazolamide, or placebo. The histograms on the left are based on Environmental Symptoms Questionnaire (ESQ-III) scores; those on the right are based on Clinical Interview (CI) scores. The group receiving dexamethasone reported a significantly lower percentage of AMS symptoms than those receiving either the acetazolamide or placebo ($P \leq .05$).

Eight of ten dexamethasone subjects, when asked which drug they preferred and while still blinded as to drugs used, preferred the drug climb to the placebo climb, whereas only three of eight acetazolamide subjects chose the drug climb.

Although subjects were randomly assigned to active drug groups and no statistically significant differences existed at base line in the study sample (Table 1), we were concerned about the possibility of confounding because a previous history of AMS appeared to be more frequent in the acetazolamide group. The climbers who reported a previous history of AMS did have higher symptom scores at the high point (AMS-C: AMS history, 1.2 ± 1.1, and no AMS history, 0.5 ± 0.5; AMS-R: AMS history, 1.6 ± 1.2, and no AMS history, 0.7 ± 0.9; clinical interview: AMS history, 2.5 ± 2.5, and no AMS history: 1.5 ± 1.5). Table 2 displays the paired differences between symptom scores on the placebo climb and the drug climb (for example, the AMS-C score while receiving dexamethasone minus the AMS-C score while taking placebo yields a mean paired difference for the AMS-C score without a previous history of AMS of -0.5 and for those subjects with a previous history of AMS of -1.2; negative paired differences indicate more favorable results). From these analyses, dexamethasone appears to be effective in reducing symptoms of AMS among all climbers, regardless of previous AMS history status. A greater degree of improvement is observed, however, among dexamethasone climbers with an AMS history. Acetazolamide use produced less dramatic and somewhat inconsistent improvements. These subgroup analysis results confirm our basic findings—dexamethasone use is effective in preventing symptoms of AMS. The small sample size of these subgroups made these estimates of symptom scores uncertain and limited our ability to ascertain statistical significance between the use of dexamethasone and that of acetazolamide because of the possible bias introduced by the slightly higher proportion of persons with a history of AMS among the climbers receiving acetazolamide.

Comments

In this study, the use of dexamethasone was effective in reducing the incidence and severity of AMS during the rapid, active ascent of Mount Rainier. The results validate our previous research, which used a different design and was complicated by the occurrence of gastrointestinal symptoms (nausea) at low elevations in the acetazolamide group.[20] The superiority of dexamethasone use reported here was not due to an adverse reaction to acetazolamide because acetazolamide-treated climbers had similar ESQ-III scores at the trailhead on both climbs. Overall, AMS-C, AMS-R, and clinical interview scores were increasingly more favorable for climbers taking dexamethasone, starting at base camp on ascent (3,000 m) and continuing to the summit or high point attained. The overall rate of AMS was significantly lower in the dexamethasone-treated subjects.

The relatively long history of safe and effective prophylactic acetazolamide is supported consistently by several studies.[4,7-17] It is difficult to explain why acetazolamide was not as effective in this study, which is similar to previous work on Mount Rainier.[13] The lack of significant differences in the acetazolamide-treated group may be due to the small sample size. In addition, a previous history of AMS was more common (but not significantly so) in the group treated with acetazolamide. Nevertheless, subjects with and without a history of AMS generally had decreased symptoms with active acetazolamide treatment (Table 2). Side effects of acetazolamide use (including paresthesia, myopia, nausea and vomiting, dysgeusia, and urinary frequency) complicate blinding and cause problems of confounding because some side effects overlap with the symptoms of AMS. If dexamethasone use is relatively more effective than taking acetazolamide, this confounding may obscure the efficacy of acetazolamide use in direct comparison where the basis for comparison is primarily self-reported symptoms.

The mechanism by which dexamethasone, a potent synthetic corticosteroid, prevents symptoms of AMS is not known. Possible mechanisms include a reduction in cerebral blood flow or cerebral vasoconstriction and improved microcirculatory integrity, which may reduce edema by decreasing filtration through the microcirculation.[18,33-35] Levine and colleagues could show no objective differences between dexamethasone- and placebo-treated groups in oximetry data, other cardiac and respiratory measurements, or indirect measures of cerebral edema in chamber-induced AMS in six subjects.[36] They did show, as we and others have, that dexamethasone use reduced the severity of symptoms (by 63%). Although we attempted to investigate how dexamethasone use might prevent AMS by searching for objective correlates with dexamethasone treatment, these attempts were unsuccessful, and thus this study does not further elucidate the mechanism of action of dexamethasone. In fact, to our knowledge the only reported objective difference associated with the prophylactic effectiveness of dexamethasone is a reduction of retinal artery dilation in a hypobaric chamber.[18] Precise, portable methods and measurements are difficult to perform in field studies during actual climbing like ours. We attempted to collect blood oxygen saturation and heart rate data with a portable pulse oximeter, but this was not feasible because of cold temperatures. Further work is needed to elucidate the mechanisms of dexamethasone because subjective, self-reported scales may well be affected by dexamethasone's well-known euphoriant effects. The absence of this euphoriant effect at low elevations, however, detracts from this hypothesis as the primary mechanism of action.[37]

TABLE 2.—*Paired Symptom Score Differences (Drug Climb Score Minus Placebo Climb Score)**

Previous AMS History	AMS-C DEX	AMS-C ACZ	AMS-R DEX	AMS-R ACZ	Clinical Interview DEX	Clinical Interview ACZ
No						
Mean	−0.5†	0.4	−0.9†	−0.1	−1.3	−0.3
SD	0.8	0.8	1.3	1.5	2.4	2.1
n	7	2	7	3	7	3
Yes						
Mean	−1.2	−0.8	−1.8†	−0.6†	−3.0†	0.8
SD	1.5	1.3	1.7	0.6	3.0	2.5
n	3	5	3	5	3	4

ACZ = acetazolamide; AMS = acute mountain sickness; AMS-C = cerebral symptoms score; AMS-R = respiratory symptoms score; DEX = dexamethasone

*Negative paired differences indicate favorable results.
†$P < .05$ compared with placebo.

The use of dexamethasone for AMS prophylaxis is not without risks, and minimal field experience to date limits knowledge of the risk. Especially worrisome are severe side effects such as acute psychosis, depression,[22] glucose intolerance,[36] and drug withdrawal,[21] the risk of which must be weighed against the benign nature of AMS in most cases. Drug withdrawal at extreme altitude could lead to fatal complications and is a distinct possibility in extreme environments, where weather and injury frequently prevent climbers from descending on schedule and may strand climbing parties for long periods. In addition, the sustained use of glucocorticoids has been shown to accentuate anoxic brain damage in animals.[38]

More work is needed before the proper role of dexamethasone in AMS prophylaxis can be defined. For now, we agree with the conclusions of previous authors[39-41] that dexamethasone use for AMS prophylaxis should be considered only for those persons without contraindications who are intolerant of acetazolamide, for whom acetazolamide is ineffective, or who undergo forced, rapid ascent to high altitude for a short period with a guaranteed retreat route.

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THE FAIRYTALE TURNS BACKWARD

I'm afraid of this lake we walk around
where even mallards on the sunny log
bring one more question, and the long dives
of gulls call to you where you stand
on the stone bridge. Some spiteful fairy's
charming you toward darker water.
Enchanted by minnows and turtles,
by creatures of puddle and pond,
you're being pulled into green algae.

No matter how many kisses I give you
you go away from me, changing into cold,
your thin blood shifting channels at night
under layers of wool and flannel. The chemicals
do what they can, but they are not magic. There is
no more magic we know of anywhere.

We live very close to the deep well. Already
you have forgotten what we were like together
and you ask me why I am crying.

We have finished eating from a single plate,
you will never sleep in my bed. I cannot
take you with me to the palace.

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